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Case Report

An atypical presentation of a case of bilateral malignant otitis externa with bilateral simultaneous facial nerve palsy

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ABSTRACT

An unusual case of bilateral malignant otitis externa (MOE) with bilateral simultaneous facial nerve palsy is presented. A case report of a 77-year-old male, known diabetic on medication, presented with bilateral otorrhoea, severe otalgia with bilateral facial nerve palsy, was diagnosed as a case of bilateral MOE, was managed with medical management followed by surgical debridement. Bilateral MOE with bilateral simultaneous facial palsy is extremely rare. The patient had severe otalgia due to which he had even 'suicidal thoughts. Due to extremely severe otalgia and bilateral facial nerve palsy, with no improvement with adequate medical management, he was managed by surgical debridement along with continuation of medications. MOE presents with otorrhoea, otalgia, granulation tissue in external auditory canal and occasionally cranial nerve palsies. Surgical debridement along with medical management in this case has fastened the recovery and resolved his symptoms.

Keywords: Malignant otitis externa, Skull base osteomyelitis, Facial palsy, Granulation tissue, Otalgia

INTRODUCTION

The term 'malignant otitis externa' (MOE) was coined by Chandler in 1968 while presenting his case series of 13 cases of temporal bone osteomyelitis. The term 'malignant' indicated the aggressive clinical behaviour, significant mortality rate and poor treatment outcome of this disease.[1] Although the term 'malignant' is controversial, as this is an inflammatory process and not a neoplasm, this term is still widely used. The suggestions are to use the term 'necrotizing otitis externa' for aggressive infection of soft tissue of external auditory canal without bone involvement, and 'skull base osteomyelitis' (SBO) when there is histologically or radiologically confirmed bone involvement.[2] Meltzer and Kelemen in 1959 were the first to describe the term 'Skull base osteomyelitis.[3] In SBO, the infection becomes invasive and the pathogen spreads to the periosteum of the temporal bone, causing necrosis. MOE usually affects the elderly and diabetic.[4] Bilateral MOE causing bilateral simultaneous facial palsy is a rare clinical presentation.

CASE REPORT

A 77 years old male, diabetic and chronic smoker was referred from a peripheral centre to this tertiary care centre with bilateral hearing loss for last five years, bilateral otorrhoea, bilateral severe otalgia and bilateral lower motor neuron type (LMN) facial nerve palsy of two months duration. On examination, he had bilateral grade IV (House-Brackmann) LMN facial palsy. His eye closure was incomplete even with maximal effort and had started to develop early stages of exposure keratitis (Figure 1A), with lower lip drooping down with persistent drooling of saliva from mouth (Figure 1B).

His external auditory canals (EAC) showed granulations in the deeper part. In the right ear, the granulations were

arising from postero-inferior wall of EAC and in left ear; granulations were arising from postero-inferior wall and anterior wall of EAC. Pain experienced by the patient was so severe that he had suicidal thoughts. His pain score on Numeric Pain Rating Scale was 10 (on a scale of zero to 10, where zero indicates 'no pain' and 10 indicate 'worst pain imaginable'). Biopsy was taken from EAC lesions and it was suggestive of granulation tissue, ruling out any malignant change. His C-reactive protein (CRP), Erythrocyte Sedimentation Rate (ESR), and total leukocyte count were grossly elevated. Pus culture from EAC showed Pseudomonas species. He was already started on with antipseudomonal intra venous (IV) antibiotics (Inj. Piperacillin+Tazobactum 4.5 g 8th hourly) from the peripheral centre for last three weeks and from this centre, Inj. ciprofloxacin 400 mg 12th hourly was added.

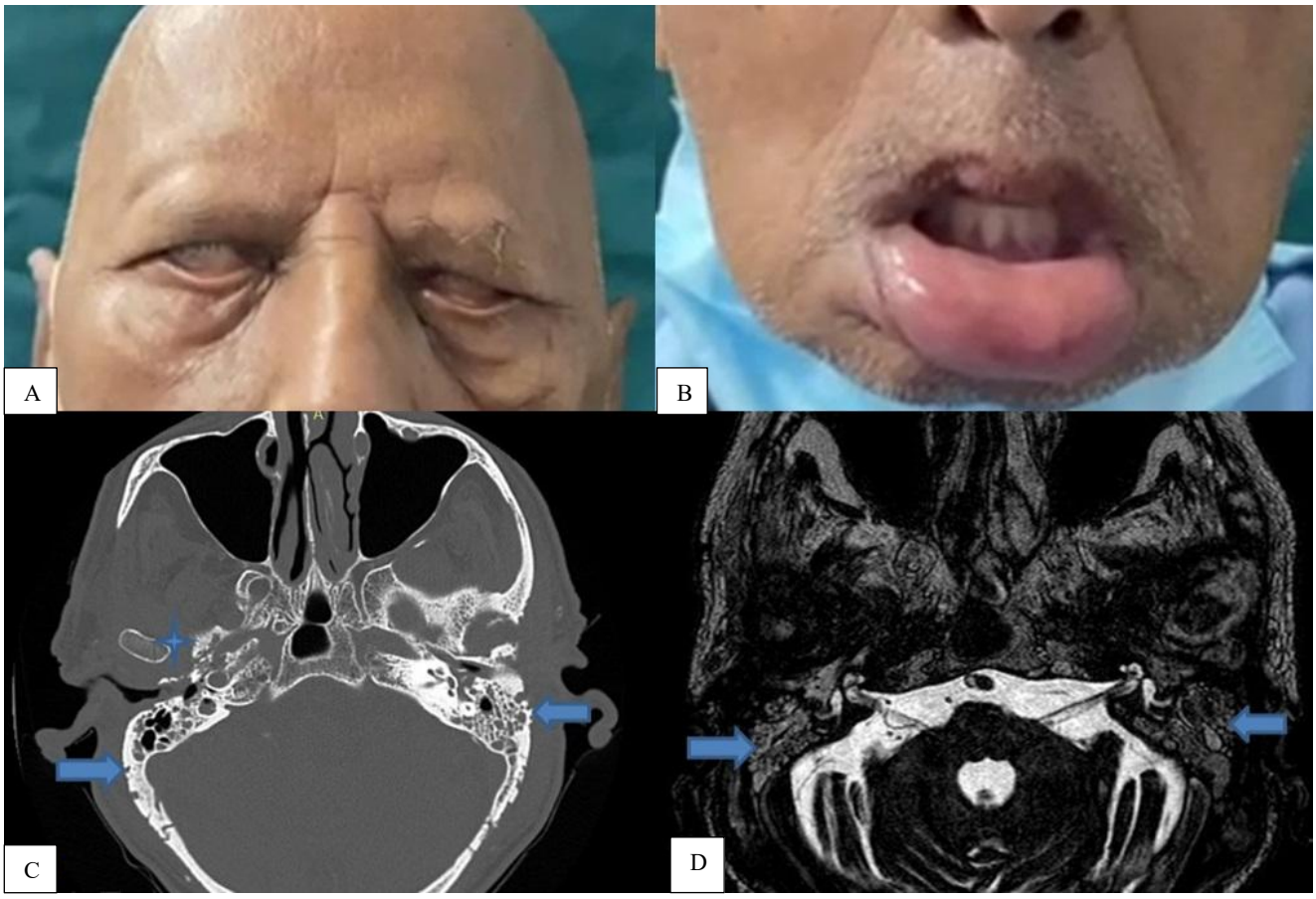

Figure 1: (A) incomplete eye closure with maximal effort and absent forehead movement, (B) drooping of lower lip due to bilateral grade IV (House-Brackmann) LMN facial palsy, (C) HRCT temporal bones showing erosion of both mastoid cortices (horizontal arrows), soft tissue filling mastoid air cells, middle ear cavity and eustachian tubes, oedema around temporo-mandibular joint (star) and (D) MRI brain and IAM showing soft tissue contents filling mastoid air cells and middle ear (horizontal arrows).

Pure tone audiogram (PTA) showed moderate mixed hearing loss in both ears with high frequency dip. Ophthalmology consultation was taken for eye care and endocrinology consultation was done for optimisation of blood sugar levels. High resolution computed tomography (HRCT) of temporal bones showed soft tissue filling both EAC, erosion of mastoid cortices on both sides, patchy erosions of anterior canal wall bone, with soft tissue contents filling bilateral mesotympanum, epitympanum, mastoid air cell system and Eustachian tubes close to the carotid canals (Figure 1C). There was dehiscence of tympanic segment of facial nerve on both sides with soft tissue around tympanic and vertical segments of facial nerve. Magnetic Resonance Imaging (MRI) scan of brain and internal auditory meatus showed soft tissue content filling EAC, mesotympanum, epitympanum and mastoid with no evidence of intra-cranial involvement (Figure 1D).

After six weeks of medical management, due to no relief in his extreme otalgia and bilateral facial palsy, he was counselled for surgical debridement. He underwent bilateral simultaneous canal wall down mastoidectomy with exenteration of all identified air cells, and bilateral facial nerve decompression under general anaesthesia in August 2022. Right ear was operated first followed by left ear, in the same sitting. On the right side, post-aural soft tissue approach was taken. The mastoid cortex was showing multiple punched out bony lesions with minimal

purulent discharge. Granulations were removed and sent for histopathological examination. All unhealthy skin from EAC were excised and tympanomeatal flap elevated. The ossicular chain was intact. Incus was removed after dislocating it from stapes head and head of malleus. Malleus head was removed and anterior epitympanum examined. Canal wall down mastoidectomy was done and the ridge was lowered till the facial nerve sheath.

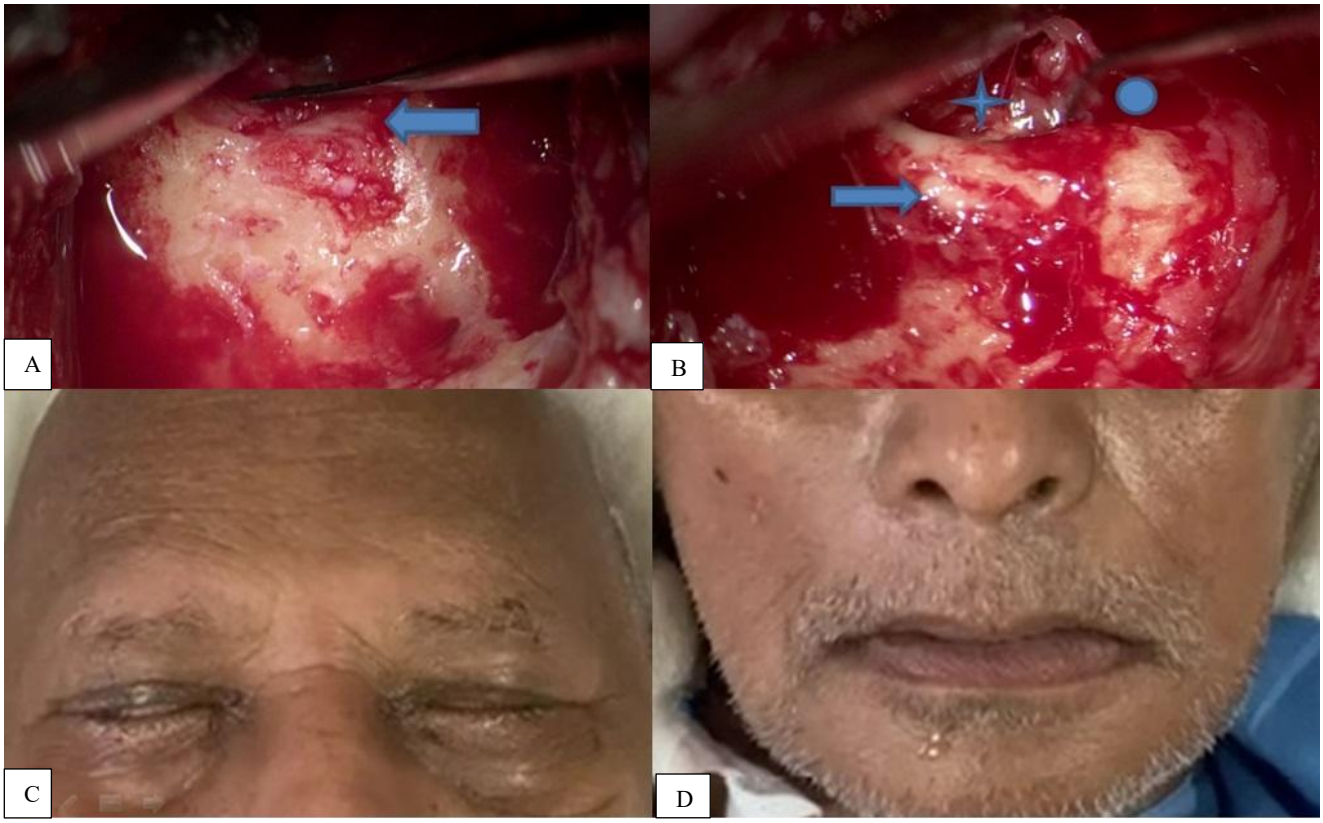

Figure 2: (A) right facial nerve decompressed 270 degrees (horizontal arrow) with sickle knife pointing to dehiscent tympanic segment, (B) left sided dehiscent second genu of facial nerve (solid circle), oedematous mucosa on promontory (star), pick pointing to stapes superstructure, and decompressed, oedematous vertical segment of facial nerve (horizontal arrow), (C) complete eye closure with minimal effort and (D) complete recovery of drooping of lower lip with no deviation of angle of mouth to either side (recovery of facial palsy to grade II).

After removing perifacial granulations, facial nerve was decompressed 270 degrees. Nerve sheath was not incised in view of actively infected field. Extensive granulations were seen around the lower portion of vertical segment of facial nerve near the stylomastoid foramen. Bony sequestrum was removed from the stylomastoid foramen region. There was nerve oedema confined to lower half of vertical segment of facial nerve (Figure 2A). Granulation tissue was removed from the Eustachian tube opening and bone at floor of Eustachian tube was drilled over the carotid canal. Patchy erosion of anterior canal wall bone was drilled, keeping temporomandibular (TM) joint capsule intact.

Similar procedure was done in the left ear, granulations from anterior canal was removed and eroded anterior bony canal wall drilled till healthy tissue was reached, keeping TM joint capsule intact. Extensive granulation tissue around the whole length of vertical segment of the facial nerve was removed. The entire vertical segment of facial nerve on left side was oedematous, which was decompressed 270 degrees.

Tympanic segment of facial nerve, although dehiscent, was normal in appearance and calibre on both sides. The decompressed vertical segment of facial nerve was covered with a piece of temporalis fascia graft. Refashioned incus was kept on head of stapes, gel foam placed in middle ear and tympanomeatal flap was repositioned after augmenting it with temporalis fascia graft kept by underlay technique. Wide meatoplasty was done on both sides and the incision wound closed in layers after placing medicated pack in the cavity. The deeper inflamed tissue obtained during surgery was sent for histopathology, bacterial and fungal stain and culture. Post operatively, he had gradual improvement in his pain scores. His pain scores on Numeric Pain Rating Scale dropped to zero at 6th week post-operatively. Sutures and cavity pack were removed on 7th post operative day. He was continued with intra-venous antibiotics for four more

weeks along with dilute acetic acid (2%) ear-drops. The culture was positive only for Pseudomonas species. His antibiotic was changed to oral ciprofloxacin 750 mg twice daily for six months. His facial palsy improved to House-Brackmann grade II (Figure 2C and 2D) at three months post-operative follow up and remained stable. His CRP, ESR and leukocyte count became normal at three months post-operative follow up. At three years of follow up, he remained disease free and pain-free in both ears with bilateral grade II facial nerve paralysis with well healed, well epithelialized modified radical mastoidectomy cavities on both sides.

DISCUSSION

The usual presentation of MOE is an elderly diabetic presenting with severe otalgia, otorrhoea and hearing loss. There may be a history of ear trauma in the form of self-cleaning of ears or syringing for wax removal. The severe deep otalgia, which is out of proportion to the clinical signs, is highly suggestive of MOE. Diabetes mellitus has a high prevalence in the reported cases of MOE, amounting to about 65 to 100% of cases.[5] Patients are usually elderly with more than 60 years of age and there is a male preponderance with a 2:1 ratio. In diabetics, the weakened immune response to Pseudomonas aeruginosa and high pH of their cerumen are identified as factors responsible for this pathology.[6] The pH of cerumen produced in the EAC of diabetic patients is higher than that of non-diabetics and this can aid bacterial proliferation.[7]

In addition, high blood sugar level impairs regeneration ability in tissue by causing blood vessel damage, and this in turn results in poor blood circulation and deficiency in immunity.[8] As the infection spreads along the skull base, several cranial neuropathy can occur and among them, the facial nerve is the most commonly affected one due to its close proximity to the EAC. If the disease spreads along jugular foramen, IX, X and XI cranial nerves can get affected and if it progresses further to the petrous apex, V and VI cranial nerves can get affected.[2] The granulation tissue in the EAC is most commonly seen in the floor and the inflammatory process is so severe that it involves the bone and changes the morphology of the compact bone of skull base. Patients usually receive intra-venous anti-pseudomonal antibiotics such as quinolones, ceftazidime and piperacillin. Change over from injectable antibiotics to oral antibiotics is usually done after six weeks. Oral antibiotics usually given are those with good bone penetration such as quinolones, linezolid, cloxacillin and minocycline.[9] While medical management remains the mainstay of treatment, in cases where there is no relief of symptoms, surgical debridement along with continued medical management has shown faster relief of symptoms, improved pain scores, radiological improvement, normalisation of inflammatory markers, and reduced total duration of treatment.[10] The possible mechanism by which surgical debridement aids in faster recovery is by removing devascularised bone and bringing well vascularised tissue in the surgical field, thus promoting healing by letting antibiotics to reach the target area. In addition to drainage of abscess, debridement of bony sequestra and acquisition of deeper tissue as specimen, surgery can aid in decompression of facial nerve in case of persistent facial nerve palsy.[11] Imaging is helpful in assessing the extent of disease and in studying the anatomical details of the temporal bone and adjacent structures. When HRCT temporal bones help to identify bone erosion and periosteal remodelling, MRI helps to identify soft tissue involvement and osteomyelitis.

Nuclear imaging studies such as fluorodeoxyglucose-positron emission tomography (FDG-PET), gallium-67 citrate scintigraphy, are used to provide supporting information. Nuclear imaging is helpful to differentiate MOE from other lesions, in monitoring disease progression, and to monitor response to treatment. Nuclear imaging was not done in this patient as he had severe otalgia and bilateral facial nerve palsy, leading to his ‘suicidal thoughts’ for which he was taken up for early surgical debridement in the form of canal wall down mastoidectomy, facial nerve decompression and subtotal petrosectomy, hoping to relieve his symptoms at the earliest. This patient remained asymptomatic for three years after surgery. According to previous studies, if the patient remains asymptomatic for 18 months after stoppage of treatment, it is considered as cured of MOE.[12]

CONCLUSION

Bilateral MOE is extremely rare and bilateral MOE with bilateral simultaneous facial palsy is even rarer. The mainstay of treatment of MOE is systemic antibiotics. However, surgical debridement has a definitive role in cases where there is no improvement in symptoms with medical management alone. Surgical debridement helps in removal of osteomyelitic bone, necrotic tissue and collected pus, which is otherwise difficult to treat by medical management alone.

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